−60°C and 4 hours at 25°C; felsite-limonite soil; and soil moisture between 0.970 and 0.975 a_w. Inhibition of growth, although erratic, was related to gaseous composition and barometric pressure, since growth occurred under Earth's atmosphere at 98 mb (*3*).

Other of our data demonstrated that the facultative anaerobe *Staphylococcus aureus* grows 2 to 3 log factors in a similar environment within 28 days; such growth was hastened by decreasing the concentration of CO_2 and increasing the barometric pressure (*4*). This organism commonly contaminates clean rooms and so must be considered a potential planetary contaminant.

Of particular interest is the observation that the diurnal temperature cycling aids in conservation of water and its availability to the microbial cell. Specifically, in simulated environments low in soil moisture and maintained constantly at 25°C, staphylococcal organisms survive with no increase in number; imposition of diurnal temperature cycling initiates growth.

Furthermore it has been demonstrated with several microorganisms that, after maximum growth is achieved, growth can be reinitiated if the soil containing the organisms is mixed with fresh soil. This fact implies that the colony can spread over a wider area than the immediate vicinity of the spacecraft.

We completely agree with the statement that water is the most critical factor for the initiation of growth in the Martian environment. As has been stated, the water near the polar cap may contain high concentrations of salt; however, it is known that facultative anaerobic staphylococci grow very well in medium containing 10 percent NaCl. Furthermore, Siegel and Roberts (*5*) have reported a bacterial ecology at still higher concentrations of salt, and Cameron *et al.* (*6*) have reported bacterial ecologies in alkaline-type soils from the Atacama Desert, Chile.

We believe that, pending further data on the moisture contents of the Martian atmosphere and soil, any attempt to modify the probability-of-growth statement expressed in the Sagan-Coleman equation is premature.

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4 AUGUST 1967

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24 April 1967

Under the conditions of the experiments described by Hawrylewicz and Hagen, the activity of water (that is, its vapor pressure relative to that of pure water at the same temperature) apparently could not fall below 0.97 at 25°C. Such experiments are of dubious relevance for Mars, whose atmosphere contains only traces of water and where the total atmospheric pressure is not much higher than the pressure of water vapor at the triple point. It is generally agreed that terrestrial microorganisms grow only in aqueous solutions. Given the dry atmosphere of Mars and the low boiling point of water on the planet (near 7°C at an atmospheric pressure of 10 mb), it seems clear that highly saline soils would provide the most favorable—if that is the word—environment for terrestrial microbes. We have discussed the implications of this conclusion [*Science* **155**, 1501 (1967)].

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Visual Adaptation: Its Mechanism

I would like to raise some objections to the evidence used by Dowling in his attempt to establish the important point that visual adaptation occurs in the retinal bipolar cells (*1*).

Dowling assumes that the rat's visual receptors are probably all rods, and the relationships presented are therefore for a rod system. I seriously doubt the validity of this assumption. Dowling's curves for the increment thresholds of the rat electroretinogram (ERG) show that the logarithm of the threshold increases linearly with the logarithm of the background over about 7 logarithmic units of adapting light. What is especially surprising is that, even when more than 90 percent of their rhodopsin has been bleached away, rat rods can still respond and that at this point the Weber relation ($\Delta I/I$ = constant, where ΔI = change in intensity and I = background intensity) still holds. This is difficult to believe when one compares rat rods with human rods since the latter obey this function over only about 4 logarithmic units above the point at which background light first begins to affect their sensitivity (*2*). Brighter adapting lights completely saturate the human rod mechanism at a concentration of rhodopsin which is only a few percent of the amount in dark-adapted rods.

Human psychophysical experiments cannot be easily compared with electrophysiological ones in the rat. Therefore, I submit the following experimental evidence to show the differences between the rod mechanisms contributing to the rat and human ERG's are also great.

Figures 1 and 2 show how adaptation to light affects the human ERG. Both the stimulus and the adapting light in these experiments cover the subject's visual field completely and homogeneously. The stimulus is a 10-μsec flash obtained from a Grass stroboscope recessed in a diffusing sphere behind the subject's head. The adapting lights, similarly placed, come from tungsten filament lamps powered by a wet-cell battery. Two chromatically different stimuli are used, one from each end of the visible spectrum and obtained by means of wave-band filters. In Fig. 1 these stimuli are balanced to have equal effects on human rods (*3*). The stimulus of short wavelengths produces only rod components, whereas the stimulus of long wavelengths elicits both rod and cone components in the ERG (*4*). In the presence of a background light of 10 mlam, a response can only be evoked by the latter stimulus. This response must result from the activity of cones since the scotopically equivalent stimulus of short wavelengths produces no response at all.

Figure 2 shows that in the presence of the same background light, even a much stronger stimulus to the rods is incapable of eliciting any components of the rod system in the ERG. The stimuli, in this case, are balanced to have equal effects on the photopic or cone receptor system of the human eye. The stimulus of long wavelengths is the same as in Fig. 1; that of short wavelengths is six times stronger than

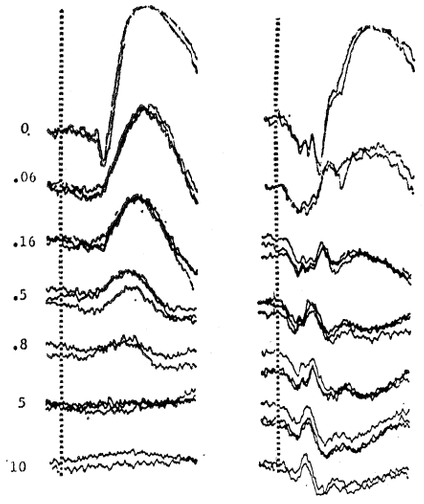

Fig. 1. Electroretinograms obtained with a short-wavelength (blue) stimulus (left) and a long-wavelength (orange) stimulus (right) in the dark-adapted state and in the presence of increasing amounts of a steady adapting light. These stimuli are scotopically balanced. The numbers on the left signify the luminance of the adapting light in millilamberts. The vertical hatched line indicates the time of the light flash. The calibration on the lower right signifies 50 μv vertically and 60 msec horizontally. Corneal positivity is an upward deflection. Two or three responses to the same stimulus are superimposed.

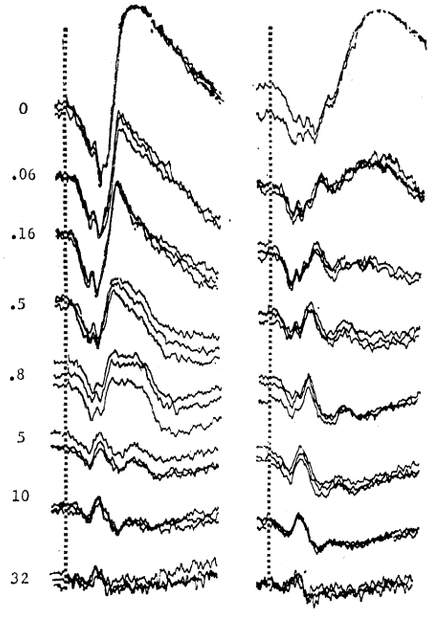

Fig. 2. Electroretinograms obtained with a short-wavelength (blue) stimulus (left) and a long-wavelength (orange) stimulus (right) in the dark-adapted state and in the presence of increasing amounts of a steady adapting light. These stimuli are photopically balanced. The numbers on the left signify the luminance of the adapting light in millilamberts. The vertical hatched line indicates the time of the light flash. The calibration of Fig. 1 also applies here. Corneal positivity is an upward deflection. Three responses to the same stimulus are superimposed.

that of Fig. 1 and about 10^5 times the threshold for eliciting a detectable response in the dark-adapted eye. I achieved photopic balance by adjusting the light energies of these stimuli to produce the same flicker fusion frequency at 30 cycle/sec. When the subject is in the dark-adapted state, the stimulus of short wavelengths produces a larger response since it stimulates the rod system much more than its photopically matched partner does. In the presence of a background light of 10 mlam, the responses to the photopically balanced stimuli became identical, another indication that with this adapting light only the cones contribute to the response.

An adapting light of about 10 mlam, which is sufficient to eliminate the rod response to relatively strong stimulation, has only a slight effect on the response of the cones, although there are 15 times as many rods receiving the stimulus. Such a light can be estimated to bleach less than 1 percent of the rhodopsin present in human rods (*2*). The response of rat rods, on the other hand, appears to make a significant contribution to the ERG in the presence of adapting lights which bleach away more than 90 percent of their rhodopsin.

Although these differences may be attributed to differences in the physiology of the rod mechanisms of man and the rat, some of the literature on this subject suggests that they may also be due to the presence of a significant number of cone receptors in the rat's retina. Dodt and Echte, in particular, have shown that light-adaptation produces a shift in the spectral sensitivity of the rat's ERG (*5*). These investigators also demonstrated that there are two mechanisms contributing to this animal's ERG flicker fusion frequency, one slow, resembling that of rods, the other faster and resembling that of cones. Sidman (*6*) has also claimed that there are cones in the rat's retina which are similar in both appearance and staining properties to those of other mammals. The ratio of rods to cones which he mentions is about 15 : 1, which is surprisingly similar to that in man. The possibility arises, therefore, that rat responses obtained in the presence of adapting lights that bleach appreciable amounts of rhodopsin are determined not by rods but by cones. Dowling's own ERG records from the light-adapted rat's eye show oscillations that resemble cone responses in the human ERG.

Determination of whether the rat retina contains only rods is important because the problem relates to generalizations about mechanisms of visual adaptation as developed in Dowling's interesting paper; it also emphasizes the difficulties that arise when it is assumed that the retina of any experimental animal consists of only one receptor mechanism.

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13 April 1967

Gouras raises an interesting question concerning the possible existence of cones in the rat retina. I do not think there are typical cones in the rat, and it is likely that the relations I discussed in my article (*1*) pertain primarily to the rat rods. However, even if there are typical cones which contribute to the rat electroretinogram (ERG), the arguments I put forward concerning the processes of adaptation and the site of adaptation are still valid. It would simply mean that the relations I discussed apply to the cone system as well as to the rod system.

The physiological evidence so far presented suggesting the presence of cones in the rat retina is mostly equivocal. As Gouras points out, Dodt and Echte (*2*) reported a small shift in spectral sensitivity (measured by the ERG) in rats adapted to light. However, the shift observed in pigmented rats is very small and is significantly smaller than that observed in albino rats. This is difficult to understand if these shifts in spectral sensitivity are due to cones. Albino rabbits are more sensitive to long wavelengths than pigmented rabbits are, and it is believed that this increased sensitivity to red light is caused by increased reflectance of postretinal tissues (mostly blood hemoglobin) in the albino (*3*). Rhodopsin absorbs about 40 percent of the incoming light in a dark-adapted rat eye (*4*) and is, therefore, a significant screening pigment. It seems likely

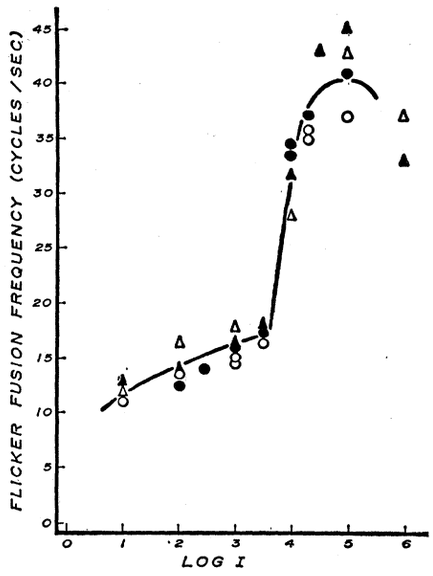

Fig. 1. Flicker fusion in the rat as a function of stimulus intensity. At low stimulus intensities, the flicker fusion frequency is between 10 and 18 flashes per second. Above log $I = 4$, the flicker fusion frequency is between 30 and 45 flashes per second. The different symbols indicate separate experiments.

to me that the small spectral shifts observed in the rat could be due mostly to a greater relative reflectance of postretinal tissue after adaptation to a strong light that bleaches away most of the rhodopsin.

In most other ERG responses evoked from the rat eye, there is no evidence of cones. For example, it is not possible to separate out rod and cone components of the rat ERG by experiments such as Gouras describes for the human ERG. Also, there is no suggestion of a cone kink or break in either the Weber-Fechner line or the dark-adaptation curve (*5*). In addition, after adaptation to strong light, which bleaches about 95 percent of the rhodopsin, the subsequent recovery of the logarithm of ERG threshold follows closely the regeneration of rhodopsin, the rod pigment, over a range of threshold of 5 to 6 log units (*5*).

The only clear physiological evidence so far presented suggesting a second visual mechanism in rats is the finding that there are two distinct plateaus in the ERG flicker fusion frequency curve. Dodt and Echte described this (*2*), and I have observed it also (*6*). Figure 1 shows the flicker fusion frequency curve in the rat. At low light intensities, the flicker fusion frequency is between 10 and 18 cycle/sec. At high light intensities (greater than log intensity = 4) the flicker fusion frequency greatly increases to between 30 and 45 cycle/sec. To identify the receptors mediating the fast flickering responses in the rat, I also measured spectral sensitivity functions in the rat at various flicker frequencies (Fig. 2). At no frequency is there a significant shift from 500 nm in the spectral sensitivity function, suggesting that rod pigment, rhodopsin, is mediating both the fast and slow flickering responses in the rat.

How, then, can we explain the two types of flicker responses in the rat? Very recently, I have observed by electron microscopy occasional inner and outer segments in the rat which appear somewhat different from the great majority of the inner and outer segments in that retina. These structures have some of the characteristics of cones (*7*) and may be the conelike receptors identified by Sidman by light microscopy (*8*). However, they do not appear to be typical cones, and they are usually not preserved very well. In the outer plexiform layer of the rat, however, we have observed occasional conelike receptor terminals. These are larger terminals than are the typical rod terminals; they have multiple invaginations and several synaptic ribbons.

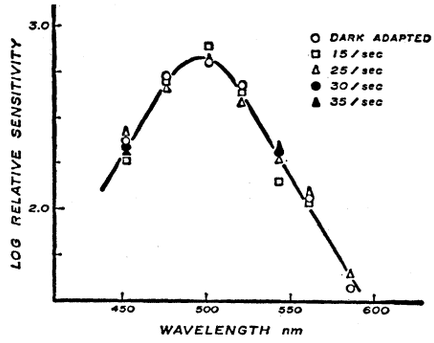

Fig. 2. Relative spectral sensitivities of the rat eye measured with flickering stimuli of various rates. No significant shifts of spectral sensitivity are observed. At flickering rates of 30 and 35 flashes per second, the measurements of relative spectral sensitivity were made at only two wavelengths.

These anatomical observations coupled with the physiological results suggest that there may be some conelike receptors in the rat retina that have rhodopsin as their visual pigment. Such conelike rods could explain the flicker fusion results described above. They may also explain how the rat retina responds over a greater range of light adaptation than the human rod system does.

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